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A review of investigations of the mosaic disease of tobacco, together with a bibliography of the more important contributions

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Concerning the nature of the mosaic disease, many important facts have been published from time to time, both in America and in Europe. However, from the published accounts of earlier investigators, especially in Europe, it is now known that various pathological appearances entirely distinct from the true mosaic have been ascribed to this disease. In reviewing the literature of the subject one is at once impressed with the contradictory conclusions reached by different investigators and the conflicting results frequently obtained in their individual experiments, which oftentimes indicate that the controls were quite as subject to the disease as plants inoculated with the virus. Unquestionably, these discrepancies to a great extent can readily be accounted for in the light of facts recently brought out by the writer (**59**, **64**)* regarding insect agencies which may become active disseminators of infection and which have heretofore been overlooked in a study of the disease.

After several years of careful investigation, Iwanowski and Polowzoff (**4**) concluded that the term "Mosaikkrankheit" had frequently been used to cover two very distinct diseases, i. e. true, infectious mosaic and "Pockenkrankheit." Mayer considered "Pockenkrankheit" simply a later phase of true mosaic and thus associated the two diseases under the term "Mosaik-

* The serial numbers in parentheses used in this paper refer to the "Index to the literature of mosaic," page 453.

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krankheit." Likewise Koning, Heintzel, and Beijerinck failed to distinguish clearly between the two diseases. The published accounts of Prillieux and Delacroix, Marchal, Goutière, Perreau, and Bouygues indicate that in many instances these investigators had confined their attention entirely to "Pockenkrankheit" or some other leaf-spot disease. This failure to distinguish the true mosaic disease from various other pathological troubles finally led to considerable confusion as to the bacterial nature of the trouble supposed to be the true infectious mosaic disease described by Mayer.

Dr. Adolf Mayer (**2**), Director of the Experiment Station at Wageningen, Holland, appears to have given in 1886 the first scientific account of the mosaic disease of tobacco, terming it "Mosaikkrankheit,"* whence the common term "mosaic" for this disease. Although Mayer failed to distinguish between this disease and "Pockenkrankheit," his accurate description of various facts and symptoms of the disease with which he worked identifies the first phase without question as the true mosaic disease affecting American tobacco. The more important facts brought out by Mayer may be briefly stated as follows:

He first proved that the disease was communicable by artificial inoculation; that the sap of healthy plants was not infectious; that an incubation period of 10 or 12 days preceded the first observable symptoms; that the disease was persistent, appearing ultimately in all immature growing parts of affected plants; that diseased sap filtered once through filter paper still retained its virulence; that sap sufficiently heated lost its virulence; and that the seed of affected plants did not carry the disease to the next generation.

Mayer failed to produce the disease in other solanaceous plants. Further, he found that liming, crowding the plants, sudden atmos-

* According to Hunger, J. H. Swieten, as a result of investigations of a disease of tobacco in the Opper-Betuwe, possibly gave the first account of mosaic in 1857. Among the tobacco growers the disease was known as "rost," a term at the present time usually applied to the mosaic disease.

In the year 1866 Swieten spent several months in Cuba in connection with problems relating to tobacco culture, but did not mention the mosaic disease as occurring here. See De tabaksteelt te Elst en omstreken in de Opper-Betuwe. Tijdschrift ter bevordering van Nijverheid, 1857, tweede reeks, Deel 5, pp. 147–167, and Beschrijving van te teelt en bereiding van de Cuba-tabak. Tijdschrift voor Nijverheid en Landbouw in Ned-Indie, 1866. Deel XII, pp. 235–316.

pheric changes, unfavorable conditions of whatever sort affecting the roots, i. e., mechanical injury, nematodes, parasitic fungi, etc., were not in any way responsible for the origin of the disease, important conclusions which several workers in the United States seem not to have known. He recognized the sporadic occurrence of the disease in the field, and finally concluded that its spread must be through the soil, both in the field and in the seed bed. He recognized the fact, however, that a transfer of the disease by means of the soil had never been proved. It is plainly indicated that Mayer held the soil in some way responsible for the origin of the disease, since he advised renewal of soil in hotbeds, crop rotation, removal of the stubs of mosaic plants, the use of chemical rather than animal manures, etc. Although Mayer did not isolate any organisms responsible for the disease, he finally concluded that it was of bacterial origin.

In 1892 Iwanowski (7) brought out additional important facts relative to the mosaic disease and confirmed many of the conclusions of Mayer. He, like Mayer, found that the sap of mosaic plants produced the disease in healthy plants and also that it lost its virulence when heated nearly to the boiling point. His results with filtered sap did not agree with the conclusions of Mayer, who stated that twice filtering through common filter paper rendered the diseased juice innocuous. On the contrary, Iwanowski found that the sap retained its virulence even after it had been filtered. In agreement with Mayer, Iwanowski also held the view that the disease was bacterial or parasitic in its nature. He did not, however, isolate any organism specifically connected with the disease, although he claims to have seen such organisms in the tissues of affected plants.

In 1894 two French investigators, Prillieux and Delacroix (8) described a tobacco trouble occurring in France, which they believed to be the true mosaic disease. Its presence was indicated on the leaves by the occurrence of spots in which they found a motile bacillus united in chains. It appears, however, that the disease described by these investigators was entirely distinct from the true mosaic of Mayer.

In 1897 Marchal (9) published a paper, "La mosaïque du tabac." He stated that the leaves of affected plants became

covered with grayish spots in the tissue of which he found a motile bacillus which imparted a yellow color to culture media. He also reproduced this disease in healthy plants by experimental inoculation. Marchal found the disease prevalent in seed beds, especially those rich in organic matter. He recommended renewal of the seed beds and sterilization as practical methods of control. The malady described by Marchal appears not to have been the true mosaic disease at all. In all essential points the disease appears to be the same trouble that was previously mentioned by Prillieux and Delacroix (8).

In 1898 Beijerinck (10) published additional facts and observations regarding the true mosaic disease. He found that diseased sap so filtered as to be entirely free from bacteria still retained its power to infect healthy plants, in this respect confirming Iwanowski (7). He showed that a very minute quantity of this filtered juice produced the disease in immature, growing tissues. He held that dried mosaic material retained its infectious properties for some time, and, likewise, that it was not rendered innocuous by remaining in the soil throughout the winter. Like Mayer (2) and Iwanowski (7), he found that heating mosaic virus to the boiling point rendered it harmless. He proved that the virus traveled considerable distances in plants but produced obvious symptoms only in immature tissues. Beijerinck claimed that the soil around diseased roots may infect healthy roots and that plants in some instances apparently recovered from the disease temporarily. Previous to the work of Beijerinck all investigators of the disease were strongly inclined to establish a bacterial origin for it, although at that time no direct proof had been obtained. Beijerinck, on the other hand, obtaining only negative evidence with regard to bacteria, advanced his theories along somewhat different lines. He assumed that the virus must be an unorganized material, fluidlike in its nature, and capable of symbiotic growth in the presence of living cells. Just what Beijerinck wished to convey by these vague and indefinite terms it is rather difficult to conclude, although it would appear as if he were foreshadowing the enzymatic theory of mosaic diseases, a theory which later was developed more fully.

Shortly after Beijerinck (10) published his results, Sturgis (16)

in Connecticut presented a careful account of the mosaic or "calico" disease as it occurs around Hartford, Connecticut, together with a summary of work done by foreign investigators up to that time. He concluded that "mottled-top" was a less pronounced phase of "calico" developing in nearly mature plants. He likewise proved conclusively by growing seedlings from the seed of calicoed plants that the disease is not transmitted through the seed. From his extended observations in the Connecticut Valley, Sturgis found that calico was very sporadic in its occurrence and that it could not be attributed to parasitic fungi, nematodes, insects, mechanical injury of the roots, cultivation, etc. He was finally led to believe that the disease was purely physiological, as the following paragraphs of his summary indicate:

(2) "The disease occurs abundantly in some localities, notably on the close, clayey soils on the east side of the Connecticut River; sparingly in other localities, where the soil is open and porous.

(7) "It seems probable that the disease is purely a physiological one, caused primarily by sudden changes of atmospheric conditions which disturb the normal balance between evaporation of water from the leaves and its absorption by the roots, and secondarily by soil conditions which prevent the speedy restoration of that balance. This supposition is supported by numerous facts."

In 1899 Sturgis (**17**) published the results of various liming and shading experiments as a preventive of calico, and as additional proof of the supposed physiological origin of the disease. In this paper he concludes "that shading may reduce the amount of calico, there seems good reason to believe"; and, with respect to liming the soil, "that the use of lime may not, in all cases, exercise the deleterious effect on tobacco that some growers suppose it to, and that there is some reason for thinking that its use may tend to decrease the prevalence of calico."

As already shown, Beijerinck (**10**), in somewhat uncertain terms, seemed inclined to place the inciting cause of the disease somewhere between parasitic and non-parasitic agencies. Sturgis (**17**) completed the step and was the first to regard the disease as a purely physiological response to particular soil and climatic factors.

He did not, however, attempt to determine the exact nature of this response in affected plants.

In the Verslag omtrent den staat van 'Slands Plantentuin te Buitenzorg over het jaar 1899 (19, pp. 73–78) the supposed bacterial nature of mosaic is discussed. Cultures of various organisms supposed to be responsible for mosaic were isolated from the tissues of such plants. These were inoculated into healthy plants, but with somewhat uncertain results. In many cases it was stated that only a slight indication of the disease followed, which, with the further development of the plants, often disappeared entirely. This was explained on the grounds that obscure conditions of one sort or another had weakened the virulence of the cultures.

It was shown that the so-called "wit kop" was simply a manifestation of mosaic. It was stated that sprinkling the virus upon healthy plants produced disease, as did placing finely cut mosaic material beneath the roots at transplanting. Sprinkling mosaic sap upon the soil ten days before transplanting, and working it into the soil did not produce mosaic.

Mosaic material was dried in the sun ten days and worked lightly into the soil. Other plots were similarly treated with mosaic material dried in the shade. It was stated that some mosaic followed each operation. In such tests, however, it may be said that there is no very certain means of determining to what extent this treatment of the soil was actually responsible for the disease until controls are taken into consideration. It is a well-known fact that mosaic may be prevalent in a field quite independently of any test.

Raciborski (19) in 1898–99 reported the results of his work with mosaic. He examined microtome sections of the leaves and stems of mosaic plants for bacteria, but found no evidence of organisms either in the cells or intercellular spaces. He determined the effects of different temperatures, exposure to the sun, etc., upon the virulence of the sap of mosaic plants. Some of his conclusions were as follows:

Mosaic sap did not lose its virulence when heated 5 minutes at 62° C. When heated one minute at 100° C. the sap still produced mosaic after 10–14 days. The virulence was lost entirely, however, when the sap was heated 15 minutes at 100° C.

When the virus was exposed to the sun in broad, shallow glass vessels for one day it was not rendered inert. An exposure for 4 or 5 days, however, destroyed its infectious nature. He concludes from this that exposure of the uppermost layers of the soil in the seed bed to the sun, will afford a practical and efficient means by which the planter can rid his soil of mosaic infection.

Mosaic sap lost its virulence when treated with potassium permanganate. Mosaic sap was treated with basic lead acetate and filtered, and the filtrate freed of lead with H_2S. Air was forced into the solution to remove the H_2S. The solution was then neutralized and sprinkled upon healthy plants. It appeared that the virulence was lost.

Water was then added to the precipitate obtained by adding basic lead acetate to the original virus. The lead was removed with H_2S, and this likewise was then driven off as before. The filtrate obtained, after filtering, was neutralized, and sprinkled upon healthy plants. The virulence was also lost.

A water extract was obtained from mosaic leaves dried in the shade. This solution was virulent when sprinkled upon plants.

Shade-dried mosaic material was then extracted with cold alcohol of 98 per cent. This extract was evaporated at 70° C. Water was added to the solid material and sprinkled upon healthy plants. The virulence was lost.

It appears that Raciborski tested the virulence of the mosaic sap following the different treatments by sprinkling it upon the leaves of healthy plants. As this method of inoculation is somewhat uncertain, however, conclusions based upon such inoculations are open to question.

In 1899 van Bijlert (**20**) mentions the occurrence of mosaic in the vicinity of the experimental station at Tandajong, Morawa. In one instance a narrow path one meter wide was a sharply dividing line between a field of tobacco which became badly mosaic and the experimental field which remained free from the disease.

From the fact that coolies did not work in the experimental field, van Bijlert is of the opinion that this largely accounts for the freedom of this field from mosaic, although surrounded on all sides by mosaic plants. Likewise, he considered that the path one meter wide served as an important means of checking the spread of the disease.

Since in the process of topping, suckering, etc., all mosaic portions removed from the plants are thrown upon the ground, Van Bijlert was convinced that the path checked the further spread of the disease through the soil by preventing rains from washing the virus of this material to the roots of neighboring healthy plants.

He considers that coolies are very largely responsible for the wholesale spread of mosaic in a field by carrying infection on their hands from plant to plant during the usual field operations of topping, suckering, etc.

Van Bijlert strongly recommended the laying out of paths one meter wide around experimental plats, not only to afford an easy means of access to different portions of the experimental field, but also to serve as an important means of preventing the spread of mosaic.

Koning (15), in 1899, largely confirmed the conclusions of previous investigators in Europe. In extensive field experiments he proved conclusively that in ordinary topping operations the disease is readily transferred from diseased to healthy plants. In fact, he claimed that in this manner as high as 88 per cent. of the healthy plants became affected with the disease. He likewise claimed that in some instances kainit and Thomas slag tended to diminish the extent of the disease.

Woods (18), in 1899, made a study of various morphological and physiological differences between healthy and discolored tissues in leaves affected with the mosaic disease and later (29) brought out certain facts relative to the disease, namely, that it is infectious, that excision of affected parts does not check its development in other parts, that the virus is generally distributed throughout the plant, and that interlacing root systems do not necessarily communicate the disease from a mosaic plant to a healthy plant. Like Sturgis (17) he concluded that the disease was a physiological response to certain unfavorable conditions. Woods went considerably farther than Sturgis, since he sought to define the actual pathological changes induced in plants as a result of this malnutrition, which he thought must involve the normal enzym activity of the plants. With regard to this point Woods (29) makes himself clear as follows:

"The disease is not due to parasites of any kind, but is the

result of defective nutrition of the young dividing and rapidly growing cells, due to a lack of elaborated nitrogenous reserve food accompanied by an abnormal increase in activity of oxidizing enzyms in the diseased cells. The unusual activity of the enzym prevents the proper elaboration of reserve food, so that a plant once diseased seldom recovers. On the decay of the roots, leaves, and stems of both healthy and diseased plants, the enzym in question is liberated and remains active in the soil. The enzym is very soluble in water and appears to pass readily through plant membranes. If young plants take it up in sufficient quantity to reach the terminal bud, they become diseased in the characteristic way."

In 1900 Heintzel (**22**), independently of Woods, came to the conclusion that oxidizing enzyms are responsible for the origin of the disease in tobacco plants.

Loew (**24**) in 1900 published briefly on the mosaic disease of tobacco. The observations of a number of practical growers in Connecticut were mentioned, but opinions were shown to be widely different as to the origin of the disease. Loew showed that an entire field may become diseased in one year, followed by a healthy crop the next season. He noted the sporadic occurrence of the disease. The oxidase and peroxidase content of healthy and mosaic plants was also compared. Many popular notions were cited which are too much at variance, however, to be regarded as established facts.

In 1900 Koning (**23**) gave a rather full discussion of his work with mosaic. Although careful examinations were made for microörganisms in the diseased tissues, all results were inconclusive.

Koning inoculated many healthy plants with soil solutions from fields where mosaic was prevalent. He was never able to produce mosaic in this way, and concluded that the virus could not exist long in the soil in an active condition. He stated that filtering the sap once through a Chamberland filter did not render it inert, but that when twice filtered its infectious nature was lost. He finally concluded that the virus of mosaic contained microörganisms too small to be retained by the pores of the filter, and that these possessed vegetative and spore-forms. Other conclu-

sions were that absolute alcohol killed the virus of mosaic, that filtered mosaic sap allowed to stand 3 months without preservatives retained its virulence. Koning states that he was unable to obtain mosaic in *Datura Stramonium*, *Hyoscyamus niger*, *Solanum tuberosum* or *Petunia nyctaginifolia* with the sap of mosaic tobacco.

Hunger (**27**) in 1902 and also in 1904 (**40**) reported the results of his observations and experiments with the mosaic disease in Sumatra He established many important facts. Although many of his experiments gave somewhat contradictory results under different conditions, he found that transplanting several times did not necessarily cause the disease to develop, although he was inclined to believe that seedlings pulled from dry soil possibly contracted the disease more readily than those pulled from wet soil. Although Hunger states that topping tobacco plants at six weeks of age produced more disease than topping them at three weeks of age, he also found that topping 1,200 plants grown elsewhere did not produce the disease in a single plant, although these plants were topped at various ages.

Hunger also grew plants from large, medium, and small seed, and concluded that medium-sized seed produced the highest percentage of mosaic plants. The large and small seed produced about the same percentage of mosaic plants. It is difficult to understand why size of seed, however, should bear any relation to the occurrence of this disease.

Hunger found that cuttings from diseased plants, whether rooted in soil or grafted upon healthy stocks, remained diseased. Many of his healthy cuttings also became diseased. He states that the trouble appeared to have no relation to fertilizer treatment. He was inclined to believe that the occurrence of the disease might be associated with extremely hot days and heavy rains.

It is interesting to note that Hunger regarded the mosaic disease as a physiological malady. He refused (**33**) to accept Wood's theory (**18**), however, that the disease was associated with the inhibitory action of oxidase and peroxidase upon diastatic action, since he maintained that these oxidizing enzyms did not inhibit the conversion of starch into sugar. He also maintained that these enzyms could not diffuse, so that plants would not be able to take them from the soil through their roots.

Hunger (36) also, in 1903, reported the results of various observations and experiments made in Deli, Sumatra, to determine how the mosaic disease was spread in a tobacco field. He showed that coolies in the operation of searching for caterpillars readily communicated the disease from diseased to healthy plants throughout the field. He stated that by touching a diseased plant first and then healthy plants "all touched plants without exception became diseased." He concludes that careless, inexperienced, and short-sighted coolies are largely responsible for the spread of the disease in a field. In this respect he confirmed the topping experiments reported previously by Koning (15).

Hunger again published, in 1903 (35), an interesting paper showing that the spread of mosaic in the Deli, Sumatra, fields is very largely due to coolies employed to search for tobacco worms. Very careful experiments were made with plats and rows, in such a way that a mosaic plant was the first plant touched. Beginning with this, only alternate plants, 3, 5, 7, 9, etc., were searched for worms. The plants represented by the even numbers were intended for controls and remained untouched. In all instances, practically every plant in the series beginning with the mosaic plant sooner or later became mosaic. At the same time, the controls, with few exceptions, remained healthy.

Hunger states that the less experienced coolies often become notorious as mosaic carriers. Coolies with this reputation are frequently troubled with defective vision as well. In the operation of worming, such coolies find it necessary to pause before each plant to determine if worms are present, and in the search the young, central leaves are handled more or less. On the other hand, the skilled, keen-eyed coolies pass rapidly from plant to plant without finding it necessary to touch the leaves in order to learn if a worm is present. Should a worm be seen it is skillfully removed. Mosaic infection under such conditions is reduced to the minimum through the operation of worming.

An instance is given of two coolies, who, from year to year, spread mosaic in all the fields they wormed. A medical examination indicated that both were very near-sighted.

Hunger (41), in 1904, also published the results of his experiments with the retransplanting of tobacco plants, before finally

transferring them to the field. This method, it seems, has been put into operation more or less generally at Deli. Experiments were made to determine what influence this practice had upon the subsequent occurrence of mosaic. The procedure was as follows: A mother seed bed *A* was prepared and sowed Feb. 17. This seed germinated Feb. 25. On the 14th of March, just 25 days from the sowing of the seed, 750 plants were transferred from *A* to bed *B* and set 3 x 2¼ inches apart. March 22, 500 plants were removed from bed *B* to bed *C* and set 4½ x 2¼ inches apart. On March 29, 250 plants were removed from bed *C* to bed *D* and set 9 x 2¼ inches apart. All were fertilized alike.

On April 2, 250 plants from beds *A* and *B* were transferred to the field. On April 8, 250 plants from bed *C*, and on April 11, 250 plants from bed *D* were set in the field.

Topping was omitted and in every way the plants were similarly treated.

The results were as follows: Plants from the mother bed *A* were healthy and reached a height of about 2¼ meters. Plants from bed *B* were not as tall and were more or less mosaic. Plants from bed *C* were very inferior in every way and badly mosaic in most instances. Plants from bed *D* were not over ½ meter in height and not a plant escaped mosaic. It is interesting to note that mosaic had appeared in beds *B*, *C* and *D* at the time of final transplanting to the field.

From these results Hunger concludes that these additional transplantings have nothing to recommend them, since they seriously interfere with the normal growth of the plants and likewise lead to the development of mosaic.

In 1904 Bouygues and Perreau (**38**) stated that they obtained, by selection, strains of tobacco resistant to the disease called by them "la nielle." The authors refer to a previous account of the disease and its symptoms by Bouygues. His careful description of the diseased plants makes it apparent that la nielle does not refer to true mosaic at all, but to some form of leaf-spot or rust.

In the same year F. Pirazzoli (**43**) published an interesting review of the literature of mosaic. She added somewhat to the confusion already existing between true mosaic and various leaf-spot diseases, since she, like Comes, called true mosaic, "Mal della bolla" and Pockenkrankheit, "mal del mosaico."

In 1904 Selby (44) reported briefly his results with the mosaic disease of tobacco in Ohio. He reviewed some of the more important practical experiments conducted by previous investigations and confirmed these results in some of his own experiments. He repeated a number of simple inoculation experiments with the virus of mosaic plants, and obtained symptoms of the disease in healthy plants after an incubation period of 9 to 12 days. He could see no difference in plants inoculated near the base and those inoculated in the tender portion. As a rule, the new growth alone showed the disease. He proved that a purely physiological chlorosis known as "yellow French" was not communicable to healthy plants by inoculation and that seed of mosaic plants produced healthy plants. The disease could not be transferred to healthy plants through the blossoms by inoculating these with the nectar of diseased plants. Contact experiments conducted with diseased and healthy plants showed an increase of 68.6 per cent. of disease in the healthy plants. In surrounding control plats the natural increase of the disease during this period was less than 3 per cent. These experiments were a further confirmation of similar experiments previously conducted abroad by Hunger and Koning.

Field observations in the Germantown district in 1903 and 1904 gave interesting results with respect to the occurrence of the mosaic disease. Considering 12 farms in the vicinity of Germantown, the percentage of diseased plants ranged from less than 1 per cent. to 43.5 per cent. On the station farm great variation was found in the prevalence of the disease in individual rows, some rows being entirely free, others showing 56 per cent. of the plants affected. Fertilized and unfertilized plats showed no difference. Preventive measures are recommended which in the main consist of the prompt removal of all diseased plants, both in the seed bed and in the field. Selby states that such diseased plants if allowed to remain become a menace to all healthy plants through the practical operations of worming, topping, suckering, etc.

In 1905 Hunger (46) further discussed the mosaic disease of tobacco. He carefully reviews the various theories of the disease, and gives a full discussion covering "Pockenkrankheit," which for

a long time was confused with "Mosaikkrankheit." Although he believed that the mosaic disease was a physiological disturbance arising from unfavorable conditions, Hunger held that an unorganized ferment of the toxophore group of Oppenheimer, rather than oxidizing enzyms, was responsible for the appearance of the disease.

In 1905 Jensen (47) published a paper in which he mentioned the different means by which the occurrence of mosaic could be lessened according to the views of various investigators, i. e., Mayer, Raciborski, Woods, Sturgis, Koning, Hunger, and others. He stated that since these workers regarded the soil in some way responsible for the origin of the disease, their attention was directed mainly to the treatment of the soil of the seed bed and the field. For the seed bed, sterilization, soil-removal, and the use of certain kinds of manures were generally recommended. For the prevention of the disease in the field, certain fertilizers were advised. Sturgis, in Connecticut, was inclined to believe that the use of shade lessened the occurrence of mosaic, Woods also considered that injury of any sort predisposed the young plants to the disease. For this reason he considered that root injury must be guarded against during transplanting, in order to reduce the amount of the disease in the field.

Jensen first reviews somewhat critically the methods employed by Sturgis, Iwanowski, and Hunger to show that the seed of mosaic mother plants produce healthy progeny. He describes in detail the results of special selection experiments which he carried on in Java with mosaic and healthy plants. He made careful comparison of the progeny of one self-fertilized healthy plant as a control and four mosaic plants grown side by side under identically the same conditions from the seed bed to the field. The percentage of mosaic plants occurring in each progeny was separately determined. Without exception mosaic plants appeared in every progeny in varying amounts.

From these experiments Jensen is inclined to believe that the progenies of the mosaic plants show greater susceptibility to mosaic than the progeny of the healthy plants, and that the careful selection of resistant races will prove to be a practical method of controlling the disease. However, from the amount of

mosaic shown by the single control progeny it seems fair to ask if Jensen's experiments have been extended sufficiently to warrant final generalizations as to the greater resistance of progenies of healthy over mosaic parents.

Delacroix (49), in 1906, reviewed rather fully previous investigations of the mosaic disease of tobacco.

As a means of preventing the disease, he advises making seed beds in new soil which has not previously grown tobacco. He also recommends crop rotation in the field, and cautions against the use of insufficiently rotted organic manure, and the choice of soils naturally too wet.

At the same time, Delacroix discusses a leaf-spot disease which he calls "la maladie des taches blanches" to distinguish it from a spot disease which he has previously described as "rouille blanche." He is convinced that the disease termed "mal del mosaico" by Comes and F. Pirazzoli is identical with his "maladie des taches blanches." Delacroix is not quite certain as to the relationship of "rouille blanche" and the "maladie des taches blanches."

Baur (50), in 1906, mentions the mosaic disease of tobacco in connection with a discussion of infectious chlorosis of the Malvaceae. He is inclined to believe that they are not essentially different in some respects. It is mentioned that the former is, however, transferred by other means than by grafting, and that the virus of mosaic is more stable, since the principle of infectious chlorosis can exist only within the living cells of the Malvaceae.

In 1907 Hunger (51) expressed his views concerning the effects of shade as a preventive of mosaic in tobacco. In a test of two plots with Deli tobacco, one shaded, and another unshaded as a control, he states that only 8 per cent of the plants became mosaic under shade, while 44 per cent became mosaic in the sun. He considers this a striking confirmation of Sturgis's results in Connecticut.

In explanation of these results, Hunger considers that shade so regulates the various physiological activities of the plant that the phytotoxin of mosaic is not generated. He contends that mosaic arises only when unfavorable external conditions stimulate the secretion of this specific toxin, which once formed has the

peculiar property of engendering a similar diffusible toxin within the cells. In some respects Hunger's toxin theory is not far removed from Beijerinck's "contagium vivum fluidum" theory.

Hunger remarks that the Deli-Sumatra tobacco, through intensive breeding, has become especially subject to mosaic. He mentions that soils most favorable to the production of the best type of Sumatra tobacco are also especially favorable localities for mosaic, while on "paja-soil" which produces the most inferior Deli wrappers, mosaic is almost unknown.

In 1908 Clinton (53), working in Connecticut, established an important fact concerning the mosaic disease. By artificial inoculation he showed conclusively that the mosaic disease of tobacco was communicable to healthy tomato plants and vice versa. This seems to be the first actual proof that this disease of tobacco is infectious to plants of other solanaceous genera.

Lodewijks (56), in 1910, reported his investigations concerning the effects of different kinds of light upon the development of the mosaic disease. By keeping the upper diseased portions of mosaic plants covered while at the same time the lower, healthy-appearing leaves were exposed, Lodewijks claims to have obtained remarkable results. Under these conditions he claims to have found that diffused light checked the disease, red light decreased it, and blue light completely cured plants of the malady. It was his opinion that an antivirus or antitoxin was thus formed in the lower, healthy leaves which destroyed or rendered inert the virus of the disease. These results are so radical that further investigation seems necessary in order to understand their meaning more fully.

In 1910 Westerdijk (57) published an account of the mosaic disease of tomato. She concludes that this disease is infectious to tomatoes, but that it is not communicable to tobacco. She likewise believes that the development of the disease is greatly dependent upon the intensity of light, strong sunlight increasing the intensity of the symptoms. In striking contrast to the mosaic disease of tobacco, she claims that the mosaic disease of tomato is carried to the next generation through seed produced by mosaic plants. Westerdijk stated that she was unable to communicate the mosaic disease of tobacco to healthy tomato plants by artificial

inoculation. Her results on this point, however, are somewhat open to question, since from the description of the plants one can not be sure that she worked with the true mosaic disease of tobacco.

Jensen (63) (1903–1911) reports his investigation of the mosaic disease of tobacco in the Dutch East Indies. A number of mosaic-free plants were selected in the field, and the progenies of these kept separate each year. By continuous selection of mosaic-free plants within these lines from 1903 to 1907, Jensen states that there was a very noticeable increase each year in the percentage of mosaic-free plants. He was led to believe that careful selection afforded an efficient means of decreasing mosaic by developing strains naturally resistant to mosaic infection.

Jensen describes and gives an illustration of a most remarkable tobacco plant found in 1899. With the exception of a single basal leaf which had developed to normal size, the growth of the leaves was limited to the development of the midribs alone. The plant was selfed but the entire progeny grown in 1910 was normal. It is very probable that this abnormal plant was an exceptionally severe form of mosaic.

Experiments were carried on to determine if excessive quantities of plant food had any influence on the susceptibility of plants to mosaic. It was found, however, that mosaic occurred quite as generally in the fertilized as in the non-fertilized plants.

Jensen (1911) states that attempts to develop mosaic-resistant lines from plants escaping mosaic infection in the field resulted in failure. At Kebon-Aroem, where mosaic is very prevalent, 93 healthy plants selected from a badly mosaic field were inoculated with the filtered sap of mosaic plants. Not a single plant escaped infection. The conclusion was finally reached that mosaic-free plants selected in a field, must be inoculated with the virus of mosaic before their resistance to mosaic can be determined.

In 1912 the writer (59) published briefly in Science additional facts concerning the mosaic disease of tobacco. It was shown that the disease is communicable to practically all genera of the solanaceous family, that the disease is dependent upon specific infection, and that simply cutting back plants does not produce the malady. It was also shown that particular kinds of aphides may be active disseminators of the disease.

In 1912 Egiz (60) in Russia published a paper concerning the growing of tobacco in southern and middle Russia. He mentions briefly the more important diseases affecting the tobacco plant in this region, i. e., rust (Pockenkrankheit), mosaic, and other diseases. He added nothing beyond what is already known concern- the mosaic disease of tobacco. Egiz considered that soil and climatic factors of one sort or another were responsible for the disease, i. e., soils too moist, soft rather than hard soils, hot sunshine together with great humidity, etc. As a means of preventing the occurrence and spread of the disease he recommended burning all diseased plants, keeping soils in the best tilth, the application of good quantities of lime, and the saving of seed from healthy plants. Egiz also mentioned another disease somewhat similar to mosaic in appearance which is not infectious and results from injuries to the roots from one cause or another. The leaves become marbled or variegated.

Chapman (61), in 1913, published the results of observations and experiments with the mosaic disease of tobacco and tomatoes. He pronounced the disease a purely physiological one, and contended that it was not of fungous or bacterial origin. He did not consider that a specialized virus was responsible for the occurrence of the disease. He regarded the theory of antagonistic enzyme action previously advanced by Woods as sufficient to explain the primary origin of the disease. Although Chapman believed that improper sterilization of infected seed beds actually increased the occurrence of mosaic in the seedlings, it is difficult to offer a satisfactory explanation for such results.

Since the time of Mayer the failure to distinguish between the true mosaic disease and various other diseased appearances has led to a literature filled with confusion and contradiction. Throughout Europe many investigators at first regarded a spot disease, "Pockenkrankheit," as a later or final phase in the development of the true mosaic disease or "Mosaikkrankheit." Others have contended that these appearances represent two distinct diseases. Throughout France especially, various leaf-spot diseases were described as the true mosaic of Mayer. Beijerinck (12) applies the term "la nielle" to the true mosaic disease. Perreau (38) speaks of "la nielle "ou" Mosaikkrankheit" and has in mind a

rust or spot disease. Delacroix (45) later designates true mosaic as "la nielle vrai."*

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INDEX TO THE LITERATURE OF MOSAIC

The following is a list, arranged in time sequence, of the more important contributions referring to the mosaic disease of tobacco or mentioned in connection therewith.

1. 1885. **Mayer, Adolf.** Over de in Nederland dikwijk voorkomende Mozaiekziekte der Tabak. Landb. Tijdschr.
2. 1886. ————. Über die Mosaikkrankheit des Tabaks. Landwirtschaft. Versuchs-Stat. **32**: 450–467. *pl. 3*. Abstract by Erwin F. Smith in Jour. Myc. **7**: 382–385. 1894.
3. 1888. ————. Heilung der Mosaikkrankheit des Tabaks. Landwirtschaft. Versuchs-Stat. **35**: 339–340.
4. 1890. **Iwanowski, D., & Polowzoff, W.** Rjabucha, bolesn Tabaka, eja pritschini i srelstwa borbi s neju. (Die Pockenkrankheit der Tabaksflanze.) 23 p. Mém. Acad. Imp. Sci. St. Pétersbourg, VII. **37**.
5. 1890. **Linhart, György, & Mezey, Gyula.** A dohány mozaikbetegsège. Mezögazdasägi Szemle, p. 1–10.
6. 1892. **Iwanowski, D.** Über die Mosaikkrankheit der Tabakspflanze. Bull. Acad. Imp. Sci. St. Pétersbourg II. **3**: 67–70.
7. 1892. ————. Über zwei Krankheiten der Tabakspflanze. Land- und Forstwirtschaft (Russisch). Abstract in Beih. Bot. Centralb. **3**: 266–268. 1893.
8. 1894. **Prillieux, E. E., & Delacroix, Georges.** Maladies bacillaires de divers végétaux. Compt. Rend. Acad. Sci. Paris **118**: 668–671.

These writers described a bacterial disease which they thought was true mosaic, but which later was shown to be "rouille blanche" (white rust).

* Terms used to designate true mosaic:

America: Mosaic; calico; gray-top; mottled-top; mottling; foxy tobacco; brindle; mongrel; chlorosis; walloon; frenching.

France: La mosaique vrai; la nielle vrai (Delacroix); la nielle (Delacroix) (Beijerinck).

Italy: Male della bolla.

Netherlands: Mozaiek-ziekte; vlek-ziekte; wit-kop.

Germany: Mosaikkrankheit; Flecken-krankheit.

Brazil (Portuguese): A molestia de "mosaico."

9. 1897. **Marchal, Émile.** La mosaïque du tabac. Rev. Myc. **19**: 13, 14.

Marchal appears to have studied "la rouille blanche" rather than true mosaic.

10. 1898. **Beijerinck, M. W.** Ueber ein Contagium vivum fluidum als Ursache der Fleckenkrankheit der Tabaksblätter. Verhandel. Kon. Akad. Wetensch. Amsterdam II. 6^5: 1–22. *pl. 1, 2.* Abstract in Centralb. Bakt. Zweite Abt. **5**: 27–33. 1899.

11. 1899. ————————. Bemerkung zu dem Aufsatz des Herrn Iwanowsky über die Mosaikkrankheit der Tabakspflanze. Centralb. Bakt. Zweite Abt. **5**: 310, 311.

12. 1899. ————————. De l'existence d'un principe contagieux vivant fluide, agent de la nielle des feuilles de tabac. Arch. Néerland. Sci. II. **3**: 164–186. *pl. 5, 6.*

13. 1899. **Breda de Haan, J. van.** Voorloopige mededeelingen over het Peh-Sem of de mozaïek-ziekte in de tabak te Deli. Teysmannia, **9**: 567–584.

14. 1899. **Iwanowski, D.** Über die Mosaikkrankheit des Tabakspflanze. Centralb. Bakt. Zweite Abt. **5**: 250–254. [Illust.]

15. 1899. **Koning, C. J.** Die Flecken- oder Mosaikkrankheit des holländischen Tabaks. Zeits. Pflanzenkrank. **9**: 65–80. *f. 1, 2; pl. 2.*

16. 1899. **Sturgis, W. C.** Preliminary notes on two diseases of tobacco. Ann. Rep. Connecticut Agr. Exp. Sta. **22**: 242–260.

17. 1899. ————————. On the effects, on tobacco, of shading and the application of lime. Ann. Rep. Connecticut Agr. Exp. Sta. **23**: 252–261.

18. 1899. **Woods, A. F.** The destruction of chlorophyll by oxidizing enzyms. Centralb. Bakt. Zweite Abt. **5**: 745–754.

19. 1899. **Raciborski, M.** Verslag omtrent den staat van 'Slands Plantentuin te Buitenzorg over het jaar 1899. 73–78; 108–110.

20. 1899. **Bijlert, A. van.** Opmerking, omtrent de verbreiding van een vlekziekte. Mededeel. 'Slands Plantentuin 43: 49–52.

21. 1900. **Goutière, J. F.** Sur quelques maladies du tabac. Jour. d'Agr. Prat. 64^1: 569–571.

22. 1900. **Heintzel, Kurt.** Contagiöse Pflanzenkrankheiten ohne Microben, mit besonderer Berücksichtigung der Mo-

saikkrankheit der Tabaksblätter. Erlangen, 46 p., 1 pl. (Inaugural-Dissertation.)

23. 1900. **Koning, C. J.** Der Tabak: Die Flecken- oder Mosaikkrankheit des holländischen Tabaks. Studien über seine Kultur und Biologie. Amsterdam. pp. 71–86, 3 fig.

24. 1900. **Loew, Oscar.** Remarks on the mosaic disease of the tobacco plant. U. S. Dept. Agr. Rep. **65**: 24–27.

25. 1900. **Woods, A. F.** Inhibiting action of oxidase upon diastase. Science, II. **11**: 17–19.

26. 1901. **Iwanowski, D.** Über die Mosaikkrankheit der Tabakspflanze. Centralb. Bakt. Zweite Abt. **7**: 148.

27. 1902. **Hunger, F. W. T.** De mozaiek-ziekte bij Deli-tabak. Deel I. Verslag van de op Deli met betrekking tot de mozaiek-ziekte genomen proeven in de jaren 1901–1902. Mededeel. 'Slands Plantentuin 63: 1–104.

28. 1902. **Iwanowski, D.** Die Mosaik- und Pockenkrankheit der Tabakspflanzen. Zeits. Pflanzenkrank. **12**: 202, 203.

29. 1902. **Woods, A. F.** Observations on the mosaic disease of tobacco. U. S. Dept. Agr. Bur. Pl. Ind. Bull. 18: 1–24. *pl. 1–6.*

30. 1903. **Bouygues, H.** Sur la nielle des feuilles du tabac. Compt. Rend. Acad. Sci. Paris **137**: 1303–1305.

A leaf-spot or rust disease is described, rather than the mosaic disease.

31. 1903. **Hunger, F. W. T.** Physiologische onderzoekingen over Deli-tabak. Mededeel. 'Slands Plantentuin 66, Hoofdstuk 5.

32. 1903. **Roux, E.** Sur les microbes dits invisible. Bull. Inst. Pasteur, Revues et Analyses, 1, No. 1.

Roux considers it probable that organisms too small to be revealed by the microscope are responsible for mosaic.

33. 1903. **Hunger, F. W. T.** Bemerkung zur Wood'schen Theorie über die Mosaïkkrankheit des Tabaks. Bull. Inst. Bot. Buitenzorg 17: 1–9.

34. 1903. ————. Een voorloopige verklaring omtrent het veelvuldig optreden der mozaiek-ziekte bij Sumatra-tabak. Tijdschrift voor Nijverheid en Landbouw in Nederlandsch-Indie **67**: 225–237.

35. 1903. ————. Het Rupsen-zoeken bij tabak in verband met het later optreden der mozaiek-ziekte. Teysmannia **14**: 632–638.

36. 1903. ————————. On the spreading of the mosaic disease (calico) on a tobacco field. Bull. Inst. Bot. Buitenzorg 17: 10–16.

37. 1903. **Iwanowski, D.** Über die Mosaikkrankheit der Tabakspflanze. Zeits. Pflanzenkrank. **13**: 1–41. *pl. 1–3.*

38. 1904. **Bouygues H., & Perreau, —.** Contribution à l'étude de la nielle des feuilles de tabac. Compt. Rend. Acad. Sci. Paris **139**: 309–310.

These investigators claim to have obtained by selection strains of tobacco resistant to the disease called by them "la nielle." This disease appears to be a rust of some sort, although it is mentioned in an abstract in the Experiment Station Record, U. S. Department of Agriculture, **16**: 677, 1905, as the mosaic disease of tobacco.

39. 1904. **Hunger, F. W. T.** Over den aard der besmettelijkheid der mozaiek-ziekte bij de tabaksplant. Handelingen, Achtste Vlaamsch Natuuren Geneeskundig Congres gehouden te Antwerpen op 24^{en} en 25^{en} September, afl. 3, p. 45–50.

40. 1904. ————————. Die Verbreitung der Mozaikkrankheit infolge der Behandlung des Tabaks. Centralb. Bakt. Zweite Abt. **11**: 405–408.

41. 1904. ————————. Invloed van het verspenen van tabaks bibit. Korte berichten uit 's Lands Plantentuin. Teysmannia **15**: 58–64.

42. 1904. **d'Utra, Gustavo.** A molestia de "Mosaico" do fumo. Boletim da Agricultura, São Paulo V. 2: 51–71.

d'Utra merely reviews the work of a number of investigators connected with the mosaic disease of tobacco. His statement that the disease rarely ever occurs in successive years is of considerable interest.

43. 1904. **Pirazzoli, Francesca.** Male della bolla e del mosaico. Bolletinno Technico della Coltivazione dei Tabacchi **3**^{4}: 1–41. *pl. 1.*

44. 1904. **Selby, A. D.** Tobacco diseases and tobacco breeding. Ohio Agr. Exp. Sta. Bull. 156: 87–114. *pl. 1–8* + *f. 1–3.*

45. 1905. **Delacroix, Georges.** La rouille blanche du tabac et la nielle ou maladie de la mosaïque. Compt. Rend. Acad. Sci. Paris **140**: 678–680.

Distinction is made here between "la rouille blanche" (white rust), a bacterial disease which Delacroix at first described as true mosaic, and "la nielle vrai" (true mosaic). He names the bacillus considered by him responsible for "la rouille blanche."

46. 1905. **Hunger, F. W. T.** Untersuchungen und Betrachtungen über die Mosaikkrankheit der Tabakspflanze. Zeits. Pflanzenkrank. **15**: 257–311.

47. 1905. **Jensen, Hjalmar.** Über die Bekämpfung der Mosaikkrankheit der Tabakpflanze. Centralb. Bakt. Zweite Abt. **15**: 440–445.

48. 1905. **Hunger, F. W. T.** Neue Theorie zur Ätiologie der Mosaikkrankheit des Tabaks. Ber. Deuts. Bot. Ges. **23**: 415–418.

49. 1906. **Delacroix, Georges.** La nielle du tabac et la "maladie des taches blanches." Ann. Inst. Nat. Agron. II. **5**: 158–205.

50. 1906. **Baur, E.** Über die infektiöse Chlorose der Malvaceen. Sitzungsber. Königl. Preuss. Akad. Wiss. 1906: 11–29.

51. 1907. **Hunger, F. W. T.** Beschaduwing als prophylaxis tegen de mozaiek-ziekte der tabak. Mededeel. Depart. Landbouw. 3: 62–68.

52. 1909. **Sorauer, Paul.** Die Mosaikkrankheit des Tabaks. Handbuch der Pflanzenkrankheiten **1**: 678–683.

Mosaic is classed as a non-parasitic, enzymatic disease. The work of Bouygues and Perreau is cited as an example of what selection for resistance to mosaic can accomplish. The fact seems to have been overlooked that Bouygues and Perreau worked with a rust disease and not true mosaic.

53. 1909. **Clinton, G. P.** Notes on fungous diseases, etc., for 1908. Connecticut Agr. Exp. Sta. Bien. Rep. **1907–1908**: 857–858. *pl. 66. f. b.*

54. 1909. **Perreau, ——.** Note sur la nielle des tabacs. Bull. Soc. Bot. France **56**: LIII–LV.

Perreau refers to the rust or leaf-spot disease previously mentioned by him and Bouygues in 1904. Perreau stated that the disease appeared on land which had not been grown to tobacco for 30 years. An abstract of these results is given under the heading "Notes on the mosaic disease of tobacco," in the Experiment Station Record, U. S. Department of Agriculture, **23**: 649. 1910.

55. 1910. **Hinson, W. M., & Jenkins, E. H.** The management of tobacco seed beds. Connecticut Agr. Exp. Sta. Bull. **166**: 1–11. *f. 1.*

These authors state that there is no evidence to show that infection arises from the stems plowed into the field.

56. 1910. **Lodewijks, J. A., Jr.** Zur Mosaikkrankheit des Tabaks. Rec. Trav. Néerland. **7**: 107–129. Abstract in Bot. Centralb. **114**: 518.

57. 1910. **Westerdijk, Johanna.** Die Mosaikkrankheit der Tomaten. Amsterdam, 19 p., 3 pl. (Mededeel. Phytopath. Lab. "Wille Commelin Scholten" Amsterdam, 1.)

58. 1911. **Russell, H. L.** Tomato breeding experiments. Wisconsin Agr. Exp. Sta. Bull. 218: 20.

Here it is claimed that the peach and cherry types are naturally resistant to the mosaic disease, and that crossing these with Earliana have given strains resistant to the disease.

59. 1912. **Allard, H. A.** The mosaic disease of tobacco. Science II. **36**: 875, 876.

60. 1912. **Egiz, S. A.** Tabakovodstvo. Glavnoe Upravlenïe Zemledi͡elïi͡a i Zemleustroĭstva, Department Zemledi͡elïi͡a, Obshchedostynnyi͡a Soobshchenïi͡a sel 'skokhozi͡aĭstvennykh Uchrezhdeniĭ i Spetsïalistov po Sel'skokhozi͡aĭstvennoi Chasti, (Russia), no. 9.

61. 1913. **Chapman, G. H.** Mosaic and allied diseases, with special reference to tobacco and tomatoes. Ann. Rep. Massachusetts Agr. Exp. Sta. **25**: 94–104.

62. 1913. **Melchers, Leo E.** The mosaic disease of the tomato and related plants. Ohio Naturalist **13**: 149–173. *pl. 7, 8 + f. 1.* 1913.

63. 1903–1911. **Jensen, Hjalmar.** Mozaiek-ziekte. Medeel. Proefsta. Vorsten-Landsche Tabak. No. 5, 1913.

64. 1914. **Allard, H. A.** The mosaic disease of tobacco. U. S. Dept. Agr. Bull. 40: 1–33. *pl. 1–5.* Ja 1914.